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Editorial Comment

All Out For Cleveland The Arrangements Committee for the Seventh Annual Meeting of the American College of Chest Physicians brings to our attention the need for making reservations promptly for the meeting at Cleveland this year. We are given to understand that desirable hotel accommodations are being rapidly taken up and that advance indications point to one of the largest attendances of any of the meetings held heretofore.

The College will have its headquarters at the Statler Hotel, which is also the headquarters for the House of Delegates of the American Medical Association. The meeting of the American Medical Association will open on June 2nd and close on June 6th. The meeting of the American College of Chest Physicians will open on May 31st and close on June 2nd.

An excellent program has been arranged and a complete report of same will be found elsewhere in this issue of the Journal. A great deal of time and effort has been expended in the preparation of the program and we know that you will want to attend these sessions.

"Information Please"—which attracted such wide attention at our meeting last year, will again have an important place on the program. Experts of national repute will appear on the speakers' platform, to answer your most difficult questions.

The latest developments in chest and allied diseases will be discussed by the leading authorities in each field of medicine.

Entertainment and good fellowship will prevail. Don't wait until it is too late, write *today* to the Housing Bureau, American Medical Association, 1604 Terminal Tower Building, Cleveland, Ohio, for your reservations.

F. W. B.

Pneumothorax Directory During the past five years, the American College of Chest Physicians has published annually, a directory of its membership. The 1941 issue of this directory *(fifth edition)* which has just come off the press, contains the names of 903 Fellows and Associates of the College situated in forty-five states of the United States, the District of Columbia, the United States Possessions, and many foreign countries.

The annual Pneumothorax Directories of the American College of Chest Physicians have been compiled and published under the supervision of the Board of Regents of the College. Effective June fifteenth, all new applicants will be required to pass an examination before they can be accepted, and have their names listed as Fellows in the annual Pneumothorax Directories of the College.

The need for these directories is apparent. Collapse therapy is used internationally in the treatment of tuberculosis and other chest diseases. Many patients lead a fairly active life. Some of them travel—others are transferred from home to sanatoria—and from sanatoria to home. These directories are designed to assist both the physician and the patient in mapping out an itinerary for pneumothorax refills. It is essential that proper collapse therapy be maintained after the initial induction. Many complications have occured because of improper induction of artificial pneumothorax. Proper training and efficiency is as essential in the administration of artificial pneumothorax, as it is in any other surgical procedure.

It is for this reason, that an organization which undertakes to publish a directory and attempts to designate those men who are qualified to administer artificial pneumothorax assumes a responsibility to the medical profession. We feel that such an organization should be an integral part of organized medicine, and that all applicants for such listings should be investigated and passed upon by a qualified board.

The Board of Regents of the American College of Chest Physicians in publishing the annual Pneumothorax Directories of the College has attempted to carrp out these provisions. We trust that the 1941 Pneumothorax Directory of the College will serve as useful a purpose as have the preceding issues.

F. W. B.

Forward With The College One of the chief objectives of the American College of Chest Physicians is the advancement of scientific programs in organized medicine.

Under the direction and supervision of the College committee established for this purpose, much progress has been made. Gaining in momentum during the past few months, many meetings have been held in various parts of this country and in foreign countries. These meetings have been well attended and an ever increasing number of physicians have had an opportunity to learn more about the modern concepts and treatment of diseases of the chest.

During the months of April and May, additional meetings have been scheduled and arranged for in widely separated parts of the United States. A few of the meetings scheduled for these months are: District Meeting, Sea View Hospital, Staten Island, New York; State Chapter Meeting, Jefferson Hotel, St. Louis, Missouri; State Chapter Meeting, Texas Hotel, Fort Worth, Texas; State Chapter Meeting, Palmer House, Chicago, Illinois; State Chapter Meeting, Atlantic City, New Jersey; and these meetings will be topped off by the annual meeting of the American College of Chest Physicians to be held at the Statler Hotel, Cleveland, Ohio, May 31-June 2nd. This is to be followed by the annual meeting of the American Medical Association at Cleveland from June 2-6th. More complete information regarding these meetings will be found elsewhere in this issue of the Journal.

Additional State Chapters will be organized by the College this year, and with efficient organization and added impetus given to this program by our College Committee for the Advancement of Scientific Programs in Organized Medicine, the continued success of this program is assured. F. W. B.

Opportunities and Responsibilities*

MATTHEW WOLL**
New York, New York

Just as gold is seldom perfectly pure, so few are blessings unmixed and even catastrophies may have a brighter side.

Wars, with their accompanying pestilence and disease have taught the medical profession many things of inestimable value to the human race. Droughts have promoted water conservation and irrigation.

World War I taught us that it is much cheaper and in fact very profitable, and also that it materially improves the fighting efficiency of our military forces, to keep the diseased unfit out of the service rather than to admit into the service both the recruit and the disease. A single case of communicable disease may work unknown havoc and misery if placed in close association with thousands of others in the military force. We have no right to mix those having a communicable disease with healthy soldiers.

The same urgency exists, with but slightly diminished force, in the case of industrial workers. An inefficient, sub-standard employee may be likened to a defective or semi-obsolete machine. Both hinder production. Both are unduly expensive and both should be brought up to their maximum effectiveness. In either case, the first step is examination and inspection.

Our own interest centers around the employee, the human machine. We can well leave the other machines—the man made machines—to the engineers and plant managers.

We now face a crisis of which no man can see the outcome. We *know* that we *must* strive for the maximum attainable efficiency in every branch of our military effort. The soldier and the worker are inseparably linked. Both are essential. This defense problem is an aggregation of many problems and there are many phases of each problem.

Today, we are discussing the health side of the Labor problem, but even this health phase has a close relationship with other phases of the Labor problem, such as wages and hours, and working conditions. All have a marked influence upon the health of the worker.

The United States Public Health Service made an exhaustive country-wide survey, not long ago, of which results were published earlier in the year. Among other interesting studies was one to determine the annual per capita volume of disability for different income groups and for various causes.

It was found that for all of the 29 causes of disability, every one of them had a higher volume in the low income groups and particularly those on relief, than prevails among those with an income of $3,000.00 per year and over.

The greatest per capita volume of disability was found to be for hernia, with about twelve and a half times as much among those on relief as among the $3,000.00 annual income groups. This was followed by tuberculosis, all forms, with nearly *nine* times as much disability among those on relief as among the $3,000.00, and over, groups.

I mention this second highest group, the tuberculous, because I will have more to say about tuberculosis later. I might add in passing that accidents caused only about twice as much disability among the lowest income groups as in the $3,000.00 and up class, and *less* than *one-fourth* as much as tuberculosis.

Our problem in Labor revolves around the question—What are we going to do about it? For the past three years there has been under consideration a nation-wide health program, aimed largely at syphilis and tuberculosis. The plan, as recommended by the Committee, of which Mr. Homer Folks is Chairman, calls for an expenditure of many millions of dollars over a period of years. Now is the time to put that program into effect, even at a time when the national budget is at its all time peak. In reality such a program will save several times its cost. It is an economy measure rather than an extravagance. It would cost millions and save hundreds of millions.

Here we are, in the midst of a program

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* Presented before the Bronx County Medical Society, December 18, 1940.
** Vice-President, American Federation of Labor, President, Union Labor Life Insurance Co.

which calls for the training of 900,000 of our young men each year for the next five years. These men will be inducted into the Federal Army and so become forever after, and unto the second generation, wards of the Government. They will be drawn largely from industry, and at the end of a year, unless war occurs, will return to industry. Roughly, ten per cent of our industrial workers will have passed into and out of the service. They will be given a reasonably thorough examination, both physical and mental, at the time of their induction and a less thorough one at discharge.

What an opportunity knocks at the door of you men and women of the medical profession. If these examinations are well conducted and the records made accessible to those who can use them to the best advantage, we will have made a very substantial inroad on the National Health Problem, insofar as it relates to diseases discoverable by such examinations.

These young men are just at the age when tuberculosis takes its heaviest toll. Also, they are at the age when such cases as are disclosed by their medical examinations, if properly conducted, are more likely to be in their minimal stage. There seems to be agreement among epidemioligists in this field that if tuberculosis is to be eradicated, it is necessary to x-ray the chests of the *apparently well* to discover the early, symptomless and unsuspected cases.

I believe that this fact has been generally admitted for many years, but the obstacle that has been insurmountable in the past, and up to a few years ago, has now been brushed aside. It no longer exists. It was the cost of making full natural size 14 x 17 x-ray plates of the chests of large numbers of people. The process of making regular x-rays on celluloid film has always been too slow, cumbersome, and expensive to be applicable to x-raying great numbers of persons. More recently, new and cheaper and more rapid methods have come into limited use and seem to have opened up new possibilities in mass x-ray examinations. Being neither a physician nor a radiologist, I do not have the temerity to comment on the relative merits of these methods—*that* I must leave to those qualified to speak. However, I have had occasion and opportunity to become somewhat familiar with the work done and the results attained here in New York City, and in nearby urban centers. This method permits the making of a thousand chest x-rays per working day at a very low cost. One of the larger and more noteworthy projects which was fostered and promoted by the Central Trades and Labor Council of Greater New York, has been the x-raying of the chests of the students in the City Vocational Schools and Trade Schools. New York City has 24 such schools, with a total enrollment of over 60,000. Of this number, about 50,000 or about 83 per cent have been x-rayed. That means that for the present at least, these thousands of young people, soon to enter our shops and offices, are reasonably free from unknown tuberculosis in any active form.

Among the older workers, those actually engaged in industry, many more thousands have been x-rayed and many hitherto unsuspected cases of active tuberculosis have been disclosed. Labor union members in this City, to the number of over 33,000 have been x-rayed. It has been estimated that every such case found, which would be cared for in a public institution, results in a saving of from $800.00 to $1,000.00 of tax payers' money. This is computed on the basis that moderately or far-advanced cases of tuberculosis cost from $1,600.00 to $2,000.00 each, to care for in a state or country sanatorium, and that 75 to 80 per cent of all cases discovered from clinical symptoms, without the aid of the x-ray, reach our sanatoria in advanced stages, whereas, of those cases picked up by the x-ray, roughly 75 per cent of them are in the minimal or early stage. Such cases entail approximately one half of the cost, both in money and time, of the more advanced cases.

These two groups, the young student of the trade and occupational high school, and the older workers in industry, have offered attractive hunting grounds for tuberculosis. But now we face, in this Country, a situation which, in spite of its seriousness, offers even greater attractions and opportunities to those most concerned with the Nation's health and with our defense program. On the one hand, we are collecting great numbers of young men in winter camps for intensive military training. On the other hand, we are assembling, in vastly greater numbers, armies of

workers of both sexes and of a wider range in ages, and we are depending on them to furnish the first army, the fighting forces, with an abundance of everything they need and at the same time to supply all of the country's civilian needs as well. In these two groups, military and civil, lie our opportunities and our responsibilities.

I think it was Dr. Dublin who said—"Folks do not die of tuberculosis. They die of neglect." If that is true, and I believe it is, whose responsibility is it to see that the tuberculous case is not neglected? There need not be long argument over that question. The neglect is seldom chargeable against the victim. The average citizen has no adequate defense against infection. Just as it is generally conceded that it is the public's responsibility to protect itself against diseases commonly spread by contaminated water supply and other disease carriers, so it is a public responsibility to protect all citizens against sprayers of tubercle bacilli. This responsibility is two-part, civil and medical. The public must supply the wherewithal and the equipment, but only the medical profession can do the job. In the Military Service, the responsibility is divided between those who must supply the facilities and the medical branch of the service which must carry on the work.

In the ranks of Labor, the responsibility must lie with those most vitally concerned. Who are they? First, the worker is concerned as a matter of self-defense. Second, the employer of Labor is concerned with absenteeism in his factory. Third, the public is concerned, both from the standpoint of self protection and, as tax payers, from the economic angle, as each case of tuberculosis which goes to a tax supported institution costs from $1,600.00 to $2,000.00. Responsibility is usually accepted when self interest dictates.

Face to face with the opportunity to do a great service and the obligation to do it, what is being done about it?

In the Military Service, it is given out that they all are being, or are to be, examined for tuberculosis by means of any x-ray of the chest, but except for New York City and Long Island, and perhaps Philadelphia, I understand that in no other large metropolitan cities have the selectees under the New Service Law been examined 100 per cent by radiograph.

In New York City, every selectee coming before the medical examination boards had a full size, 14 x 17 chest x-ray made on paper film by the method that has been in use so extensively for the past eight years. First returns of findings come from Queens County. There in the 104th Field Artillery Armory in Jamaica, all selectees of Queens, Nassau, and Suffolk Counties were examined and x-rayed. Of the 652 examined, previous to November 30th, 18 or 2.75 per cent were found to have x-ray shadows, suspicious of tuberculosis or in need of further check-up.

If perchance these first returns should later prove to be representative of the whole country, it would mean that the x-ray examination of 900,000 men each year for five years would bring to light some 124,000 new or unsuspected cases of tuberculosis or possible tuberculosis.

It is important to remember in this connection that all of these 18 men whose x-rays pointed the finger of suspicion at their lungs, had already been given a preliminary medical examination by their local draft board doctors previous to coming before the induction board. What a blow it would be to the spread of tuberculosis in our Country if a hundred and twenty-four thousand unknown cases and unsuspected cases of tuberculosis were revealed to the medical profession for their attention through the x-raying of the chests of all recruits for the next five years!

But, so far I have seen no public announcement to the effect that the Medical Services of our Military establishment have even begun to x-ray either soldiers now in the regular army, National Guardsmen inducted into the Federal Service, or selectees under the new law. All x-ray examinations made to date, have, I understand, been made by agencies other than the military medical service. For example, here in the Bronx, at the 105th Field Artillery Armory, the x-raying was done by our own City Department of Health, under the supervision of Dr. Herbert R. Edwards, Director of the Division of Tuberculosis.

X-ray equipment for performance of this service is said to be "on order." In the meantime, the new recruits are being inducted into an army that has not been examined for tuberculosis. I leave it for you doctors to say

whether such a course is justifiable and whether it is conducive to the elimination of tuberculosis from military forces.

An interesting item recently appeared in the newspapers. The following is from the *New York Times* of October 18th, 1940:

FORT DIX DIVISION DROPS 15 OFFICERS

Col. T. F. Voeller Among Those Relieved of Duty After New Physical Examinations

ACTION A BLOW TO SOME

Several Are Reported to Have Sold Businesses Before They Were Inducted Into Army

FORT DIX, N. J., Oct. 17—Fifteen ranking officers of the Forty-fourth Division, including a regimental commander, an executive officer and a major, were relieved of duty with the division today because of physical disqualifications.

Although all of the officers were subjected to intensive physical examinations before they were inducted into the Federal Service, they did not receive chest x-rays or electrocardiograph tests until this week, when more thorough examinations were given. It was reported that twenty-seven officers were to have been relieved, but the entire group was reexamined yesterday at Fort Jay Hospital, Governors Island, and only fifteen of that number were finally disqualified . . . .

This is as striking a demonstration as one could wish of the urgent necessity for including a chest x-ray as a routine part of the medical examination of every person in the Army, Navy, Aviation Corps, Marine Corps, and every other branch of the defense service from the highest to the lowest in rank.

Fortunately, there is no insurmountable impediment to the accomplishment of this end. It is being done in England, in Canada, and in Australia. It can be done here. It need not occur again that a division of National Guard be inducted into the Federal Army without an examination adequate to determine their fitness for military service. It need not happen again that our National Guard units be officered by men not physically fit for military service. This result of the x-ray examination of the officers of the Forty-fourth Division raises the question—How many other divisions now in the Federal Army have fifteen officers who should be in sanatoria instead of in barracks?

Coming back, in closing, to the opportunities and the responsibilities in the realm of Labor in respect to controlling and ultimately eliminating tuberculosis as a health menace; Labor as the service arm of our defense program, and particularly organized Labor which is far more articulate and in infinitely better position to act and to accomplish, is keenly alive to the need for an active, aggressive, concerted drive against disease, because we are primarily interested in the well being of the worker. An essential of well being is being well. Our opportunities measure our responsibilities. If there is no way in which we can help to improve the health and therefore the effectiveness of our two armies—the fighting army and the army of supply—then there is no responsibility. But, if we have now an opportunity to be of service in that field and fail to embrace it to the fullest extent, then the responsibility is upon us and it is a heavy one. We must not fail.

570 Lexington Avenue.

Recurrent Spontaneous Pneumothorax

HENRY G. HADLEY, M.D., F.A.C.C.P.
Washington, D. C.

Spontaneous pneumothorax is due to the entrance of air into the pleural cavity which is usually from the lung, but occasionally from other organs of the thorax. It may be caused by any abnormal condition of the lung, the pleura, or the mediastinal organs. When pneumothorax occurs where tuberculosis, infective processes or tumors which destroy lung tissue are causative agents,[1] the clinical picture of the primary disease predominates. In asthma and chronic emphysema, spontaneous pneumothorax may occur

due to atrophy and distention of the lung tissue.[2]

While a large proportion of spontaneous cases are of the benign type, the label of tuberculosis should not erronously be applied with all its implication. Still, it is more tragic to dismiss a case as benign and insignificant and later to find advanced tuberculosis. Pneumothorax is occasionally the initial symptom leading to the diagnosis of tuberculosis, being also quite frequent in the advanced stages of the disease where it leads to disastrous results. In pulmonary tuberculosis where a rupture of a necrotic subpleural tubercle produces a communication between the bronchi and the pleural cavity, a serious pleurisy follows from infection of the pleura. Where a superficial cavity ruptures and evacuates into the pleura, a pyopneumothorax and often a lung fistula may result. While fistulas in tuberculous tissues are bronchopleural rather than parenchymatous, they are often bilateral and lead to pleural infection. In both the benign and tuberculous forms, the left lung is more often affected than the right.

The form occurring in apparently healthy individuals was first named and described by Itard in 1803.[3] This non-surgical form occurs most often in children and young adults. While Laennec[4] accurately described this condition, its tuberculous character was first described by McDowel[5] in 1856, but the name "pneumothorax simplex" was suggested by Kjaergaard.[6] Previous to Kjaergaard, pneumothorax was considered to be an evidence of tuberculosis, although increasing evidence demonstrated that apparently healthy individuals such as college students were affected.[7] Leggett, Meyers, and Levine[8] found that more than fifty per cent of their series had negative Mantoux tests. This affects individuals who previously appear to be in perfect health and in whom the most careful examination does not reveal a cause, although it appears more often in the underweight.[9] These benign afebrile cases usually do not develop an exudate and spontaneously recover. While the pneumothorax is often preceeded by some kind of mechanical strain, in many instances the causative action is trivial or undetermined.

While in children, the prevailing cause is pneumonia and in the adult, the prevailing cause is tuberculosis, in a large group, it is idiopathic. The condition may follow the rupture of valve vesicles or congenital lung cysts. Scar tissue vesicles are formed by cicatrical changes in the apices, the base of which have a valve-like structure which causes them to distend and finally rupture. They may also form without scar tissue where there are local emphysematous changes or congenital lung cysts. These valve vesicles may be found either in one lung or both lungs and may be found by x-ray examination in rare cases where the walls are thick,[10] or by thoracoscopy. Pneumothorax simplex is found more frequently in men between the ages of 20 and 40 years and the outlook is good except in cases of hemopneumothorax from internal hemorrhage and in bilateral or tension pneumothorax.

Hemothorax following pneumothorax, due to the tearing of adhesions, is very serious and may cause death from intrapleural bleeding and resulting pressure.[11] Pneumothorax may follow accidents or pulmonary embolism and has been reported in several instances following positive pressure, intratracheal anesthesia with several ending fatally.[12] It sometimes follows laparotomy and other surgical procedures.[13] Syphilis[14] and silicosis[15] are occasional predisposing factors. Spontaneous pneumothorax may occur from a rupture of neoplastic ulceration of the esophagus, bronchi or of a subdiaphragmatic abscess. The alternating form occurs where valve vesicles are present in both lungs, these rupturing at different times.

Cases having from eleven to fourteen recurrences have been recorded.[16] The atypical types are the recurrent, the more rare, being the chronic form. Spontaneous pneumothorax may recur so readily, especially where the pleura is free and air absorption rapid, that the subject may develop a pneumothorax phobia. These recurrences are due to subpleural bullae, sclerosis of tissues, congenital malformations and congenital emphysema especially in the familial types. Bachmann[17] records a family in which both the father and daughter suffered recurrent pneumothorax. Congenital abnormalities and deficient pulmonary resistance are common in asthmatic families.[18] It is difficult to distinguish differences between congenital or acquired cysts, the rupture of which causes

pneumothorax. Multiple cysts cause the radiographic appearance of the "honeycomb" lung. Alterations of the pulmonary artery are especially prominent in congenital lung defects.[19] The bilateral form may be simultaneous, successive, or alternating. In the chronic form, the air cavity remains at a standstill, although the possibility of a lung cyst or a localized atelectasis must be ruled out.

While children may have pneumothorax from early respiratory effort in the first two weeks of life, some of these may be due to obstetrical trauma,[20] congenital malformations,[21] subpleural emphysematous or congenital vesicles.[22] A large air-containing space in an infant's chest may be a spontaneous pneumothorax, a congenital pulmonary cyst or a congenital diaphragmatic hernia. A positive pressure pneumothorax in an infant causes acute respiratory embarrassment and may be fatal if not relieved promptly by aspirating air. Infection of the pleural cavity is very common where it follows pneumonia, and is inevitable after the rupture of an abscess. Collis and Foster-Carter[23] described a case of spontaneous pneumothorax in an infant of eight weeks in which autopsy revealed a rupture of a staphylococcus lung abscess which had the radiographic appearance of a pulmonary cyst.

The cause of spontaneous pneumothorax is ascribed as a rupture of subpleural ampulla vesicles which may be of either scar tissue or emphysematous origin.[24] Blebs of the scar tissue type, which predominate at the apex, are fluid-containing in the beginning and produce secondary scar and adhesion formation. The emphysematous type are air-containing, and rupture because of increased intrapulmonary pressure. Rupture rarely occurs on the posterior surface of the lungs as the cause is largely mechanical. The front and axillary portions have more respiratory motion than the posterior because of the rib attachment to the spinal column. This is more pronounced when the person lies on the back, which may account for the cases which occur during sleep. The left side is more often affected probably because the heart produces more pulmonary motion on that side.[25] Some cases arise from mediastinal emphysema. This requires an extension of interstitial pulmonary emphysema to the mediastinum. Distention of the mediastinal tissues produces severe substernal pain radiating to the neck and shoulders, giving a sensation of substernal pressure. There are no constitutional symptoms. Physical findings are subcutaneous emphysema in the neck, a decrease in or obliterated heart dullness and x-ray evidence of air in the mediastinum.[26] It is remarkable that even in the chronic forms with the presence of bronchial fistula, effusion usually does not occur.

The characteristics are a sudden illness occurring usually in males of 20 to 30 years who are otherwise in perfect health, without fever and with no effusion or only an insignificant quantity. In the majority, there is no sign of tuberculosis. They heal spontaneously in about four weeks and, as the gas is re-absorbed, no evidence of any pathological process is found.

In the benign cases, the degree of collapse is often limited by the closure of the pleural opening as the collapse itself contracts the surface area, and in tuberculous cases, the limitation is often the result of pleural adhesions. Tuberculosis does not appear after spontaneous pneumothorax any more frequently than in the general population, nevertheless, every investigation should be made into the history of malaise, loss of weight and hemoptysis.[27] The sputum and feces should be examined for tubercle bacilli. Temperature records, sedimentation rate and x-ray studies should be made of all cases.

The benign forms due to the rupture of subpleural bullae are afebrile with rapid absorption, absence of serous reaction, and conservation of the general health. Bullae may be demonstrated in the roentegenogram after re-expansion,[28] and are found at autopsy.[29] They follow areas of localized emphysema and the rupture of an alveolus, allowing the air to escape into the interstitial tissue. The constant gliding of the pleural layers thins the bleb so that it ruptures upon coughing or exertion. Complete collapse is more common in the benign form, as the pleural aperture remains open until the lung is completely collapsed. Re-expansion is delayed until the pleural lesion heals and in a few cases does not occur at all. Progressive or valvular pneumothorax is due to a flap of pleura which allows air to enter during inspiration and closes during expiration. The

intrapleural pressure, after complete lung collapse, rises above atmospheric pressure, causing displacement of the heart and mediastinum.

The elasticity of the lung progressively diminishes from the periphery to the hilar regions which are almost devoid of elasticity. The velocity at which the air is introduced into the pleural space is a deciding factor of rupture of the mediastinum and consequent production of successive bilateral pneumothorax.[30] The vital capacity is the greatest determining factor in relation to the amount of air in the pleural spaces necessary to produce respiratory embarrassment. Dyspnoea is not due altogether to the diminished vital capacity from the amount of air introduced but to the shift of the mediastinum to the opposite side which hinders the aspiration respiratory factor and interferes with the proper venous return to the right side of the heart. The younger individual with more flexible mediastinum, tolerates pneumothorax poorly.[31]

The intrapleural pressure is the difference between the elastic tension of the normal lung and the atmospheric pressure. This is normally negative except in the first few days after birth.[32] The measurements are between −5 and −10 mm. of Hg. in quiet expiration and inspiration respectively, and may reach 30 or 40 mm. on very deep inspiration, differing with altitude and posture. The dorsal recumbent position is less than the upright due to the lower intrathoracic volume. This difference may amount to 10 cms. of water in the open type of manometer. In pneumothorax, the contralateral lung does not remain indifferent to pressure variations on the pneumothorax side or the mobility of the diaphragm in inspiration and expiration. The mediastinum is not a fixed immobile structure, but moves, not only with respiration, but also with extrinsic vector changes.[33]

The onset sometimes follows an accident or physical exertion, but frequently occurs without any such cause. The occurrence of a spontaneous pneumothorax may be unnoticed and detected incidentally, or may have sudden severe onset. The most common symptom is pain which involves the entire side of the chest or is localized in the shoulder or back. As the acute onset often may simulate an acute cholelithiasis, a renal calculus, angina pectoris, or perforation of a peptic ulcer, it should be suspected in any acute pain in the chest, upper abdomen or back. Dyspnoea is common and may be severe at the onset, gradually decreasing, or slowly developing. Usually there is marked pallor. Cyanosis and a sharp rise in temperature are quite frequent in children, but both are exceptions in adults. The pulse rate is always very high. Cough is frequent, but not constant and mostly of a dry, unproductive type.

In tension cases, when the perforation acts as a valve, there is a sudden pain in the chest with dyspnoea, tachycardia and general symptoms of shock due to excessive pressure in the pleural cavity. This is more apt to occur if the pressure is increased by coughing or by forced expiration due to severe pain. If chills and fever are present, it may be confused with pneumonia and where rigidity of the upper abdomen is present, the condition may simulate perforated peptic ulcer. The most important physical signs are an immobile expanded chest with limitation of respiratory movements and dilatation of the intercostal spaces on the affected side. There is hyper-resonance or tympanitic percussion note, absent breath sounds and absent or diminished vocal fremitus, lowered diaphragm and displacement of the mediastinal contents to the opposite side. Frequently, tinkling musical rales can be heard, but the coin sound is not always found. The apical heart beat is deviated or completely absent. The heart sounds are fast, but regular, unless there is an accompanying cardiac disorder. X-ray examination and aspiration of air after paracentesis prove the diagnosis.[34]

The acute abdominal form simulates gall bladder, appendix disease or a perforated peptic ulcer with vomiting, rigidity, rapid pulse and characteristic facial expression. There is great variation in the intense localization and radiation of the pain.[35] Pain in some degree is invariable, but even in minor cases is slight. Syncope or convulsions at the onset are relatively rare. The anginal form simulates angina pectoris with severe precordial pain, radiating to the left arm with symptoms of shock. In the progressive valvular type, all symptoms may be severe and the patient rapidly passes into a stage of acute shock with orthopnoea, cyanosis, progressive cardiac failure and collapse.

The occurrence of pyopneumothorax is almost pathognomonic of pre-existent pulmonary disease, most often tuberculosis, as the benign form rarely forms more than an appreciable amount of fluid. Where there is a persisting perforation or recurrent attacks, one therapy is to produce an aseptic obliterative pleurisy. Differential diagnosis is only made certain by roentgenology and even then, a period of observation may be required before the etiology may be established.

The immediate outlook in each case depends on the relief of the initial shock and the proper treatment of the progressive valvular type, while the subsequent progress is greatly influenced by the presence of purulent effusion or the formation of a bronchopleural fistula. In such a case, the first requirement is to relieve shock and collapse. In the benign type with either partial or complete collapse, rest in bed is necessary until all symptoms have disappeared and the lung gives evidence of continuing expansion. The prognosis is usually good, but as chronic and alternating cases are apt to occur, the possibility must be considered. The symptoms vary according to the extent of lung collapse and consequent mediastinal displacement. Dyspnoea is variable while fever is usually present. The cough is short, dry and hacking resembling that of early stages of pleurisy. Sputum may be present and is occasionally blood stained.

If tension pneumothorax exists, prompt and energetic action may be life saving. A pneumothorax apparatus for pressure readings and measurement of air aspiration is useful, but if an emergency exists from excess intrathoracic pressure, a hypodermic needle of sufficient size will give relief of the symptoms due to mediastinal displacement by allowing air to escape until the intrapleural pressure is reduced. The needle may be retained in place for several days and connected with a water apparatus to measure the evacuated air, leaving it until the air ceases to escape,[36] or a Cope self-retaining cannula with a mechanical valve arranged by a rubber finger stall with a slit in the end. Marriott and Carter[37] connected the pneumothorax needle with a suction apparatus adjusted to a negative pressure of 2 cm. of water and left this for 12 to 24 hours until air ceased to escape. The disease may be shortened by aspiration of air. Bilateral pneumothorax requires bilateral puncture and aspiration. This is performed on the side in which the pneumothorax first appeared.[38]

Where a spontaneous pneumothorax becomes chronic, artificial pneumothorax to collapse the lung more completely may hasten healing of the pleural opening or gomenol in oil injections may produce adhesion of the pleural surfaces.[39] Salt solution and other chemical agents have been used with a varying degree of success.[40] Where a permanent fistula is present, thoroscopy or operation and cauterization of the fistula has been successfully accomplished.[41]

Where serous effusion causes dyspnoea or continued fever, aspiration is indicated. If the pleural cavity is purulent, aspiration and negative intrapleural pressure may aid lung re-expansion. Where the pus is thick, washing may be necessary. In some cases, closed drainage may suffice, while in others, continuous suction or open drainage followed by thoracoplasty may be necessary.

Consecutive Cases Occurring In Office Practice

Case 1—L. F. N., male, white, age 21, was first seen September 19, 1939. The chief complaint was severe pain in the chest with vomiting and rigidity of the upper abdomen. X-ray examination demonstrated a spontaneous pneumothorax of the left side. Recovery was uneventful.

This was a case of idiopathic pneumothorax which simulated a perforated peptic ulcer.

Case 2—Faith E., female, white, age 2 years, was first seen on February 3, 1933. Physical findings were: temperature 104.6, an area of consolidation in the lower left lobe and a large tympanitic area above. X-ray examination showed a lower lobe pneumonia with pneumothorax above. Recovery occured without further complications.

This was a case of spontaneous pneumothorax secondary to lobar pneumonia.

Case 3—Mrs. N. M. K., white, age 42, was first seen on October 5, 1934. Physical and x-ray studies showed a right sided hydropneumothorax without any demonstrable cause. This gradually absorbed, but one year later, the fluid re-accumulated. A diagnosis of adenocarcinoma was made by a biopsy of superficial gland which appeared in the groin,

later x-ray examinations showing a tumor of the pleura. Death occurred on November 16, 1939. An autopsy was not performed.

This case was pneumothorax secondary to a malignant disease.

Case 4—H. P., male, white, age 54, was first seen on June 9, 1936 after a fall from a ladder striking the chest on the left side. Physical examination showed spontaneous pneumothorax on the left side of the chest while x-ray examination revealed multiple lung cysts with pneumothorax. The condition never entirely cleared up and pneumothorax is still present after more than four years.

This was a case of chronic pneumothorax due to rupture of lung cysts.

Case 5—W. C. U., male, white, age 28, was first seen October 3, 1935. He was a chain store manager and while reaching for a package on a high shelf had an attack of increasingly severe pain in the left chest. Examination revealed complete leftsided pneumothorax. He was hospitalized for two weeks. Tension pneumothorax developed which required a puncture to relieve the pressure.

On July 10, 1937, he had a similar attack on the right side which produced milder symptoms and from which he recovered more rapidly. On February 18, 1940 he had a third attack on the right side and a fourth attack affecting the left side, came on April 6, 1940, both of which were partial pneumothorax. Careful physical examinations and repeated x-rays revealed no pulmonary lesions.

This case is the recurrent type of pneumothorax.

Conclusions

Pneumothorax appears more often than is usually recognized. Careful physical examination and x-ray study should be made of all suspected cases, as the symptoms may be so mild that it would not be detected, or the onset so severe as to be confused with a coronary attack or a perforated viscus as in case one. It also may be secondary to other more serious lesions as in cases two and three.

So called benign pneumothorax may not always result in spontaneous cure. Tension pneumothorax may develop in any case, the pressure of which must be relieved in order to save life. This especially is the case if it is bilateral, or if an intrapleural hemorrhage occurs. Some cases become chronic where cysts are present or bronchial fistulas develop as in case four. Recurrence is frequent where defects or congenital malformations are present. Recurrent forms may be present in patients without demonstrable cause as in case five.

1252 Sixth Street, S. W.

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Clinical Aspects of Hilum Tuberculosis in Children

LEO V. SCHNEIDER, M.D., F.A.C.P., F.A.C.C.P.
Glenn Dale, Maryland

Hilum tuberculosis or bronchial gland tuberculosis is now definitely recognized as a clinical entity observed in children and diagnosed as primary infection. The diagnosis, however, is not an easy task. In children, physical signs are usually less demonstrable than in adults, even in those cases where we are dealing with lesions of adult type. Rales of any considerable value are heard only when the disease has progressed to an advanced stage. In young children we deal mostly with latent tuberculosis of tracheobronchial lymph nodes, called hilum tuberculosis, while in older children up to the second decade of life apical involvement may indicate the presence of an adult type of infection. In early childhood the lymph nodes which function as filters localizing any infection, are more efficient than at any other period of life.

It has been shown that tubercle bacilli can live within lymph nodes for a long period of time without demonstrating any appreciable clinical signs. The nodes become enlarged but the process is localized, calcification takes place, and thus extension of the pathological process is prevented.

There is still a belief that the tuberculous infection in human beings is almost universal. The fact that a comparatively small percentage of individuals develop clinical disease, brings to fore the constitutional resistance, the question of allergy and immunity in human beings toward tuberculosis and many other theories very plausible as far as discussions are concerned, but not sufficiently clear to explain the nature, extent and limitations of these biological and physiological factors. Constitutional differences in human beings in regard to tuberculosis are un-

deniable. We know that some people are more susceptible to the disease than others, the chances of exposure being equal. Then again, any abnormal condition of the individual such as infections of the upper respiratory tract, nasal obstructions, infected tonsils, hypertrophied adenoids—all these conditions by lowering the resistance are liable to create a greater susceptibility to tuberculosis.

Most children become infected with tuberculosis in infancy, the most susceptible period being the second and third years of age. In regard to the school age children, there are many special factors which we feel may favor the development of tuberculosis. Among them are poor health habits, improper diet, neglect of upper respiratory infections, too strenuous and poorly directed social life, late hours, outside work after school hours, and others.

In early childhood the infection shows its first clinical manifestations in the hilum glands. If the child is further exposed to massive doses of infection, the lesion may show a tendency to spread upward and outward into the parenchyma of the lung. When that occurs the infection then may be recognized as pulmonary tuberculosis of the adult type.

However, we can not say definitely that the lesion is always limited to the hilum. We have seen almost every form of pulmonary tuberculosis in early childhood and adolescence, and while we would say that the primary infection occurs predominantly as hilum lymph nodes tuberculosis, the lesion seen in the older children is frequently of a latent indolent type of infiltration that causes little or no disturbance in the general physical condition.

Valuable information concerning the tracheobronchial lymph nodes infection may be gained at times by means of physical examination, search being made especially for impaired percussion note over or near the upper portion of the sternum and to the right and left of the spine. However, this is not sufficient and not always reliable. We can not base our diagnosis on physical examination only. It is not a practical measure of case-finding in the early latent primary infection in children. Physical examination may help to select those whom we suspect and whom we would like to study further. However, final diagnosis will depend on consideration of all the following factors:

1. History.
2. Symptoms.
3. Physical signs.
4. Tuberculin test.
5. X-ray evidence.

In regard to history, we are particularly interested in knowing whether the child has been intimately associated with any person who has had pulmonary tuberculosis. The closer the contact, the more chances there are for infection. Prolonged exposure of a child to an open case of pulmonary tuberculosis will surely result in an infection. And an excessive infection will produce disease.

In reference to the symptoms, we have said already that many children who have the primary type of tuberculosis may not manifest any symptoms at all referable to the disease, even the progressive lesion. However, the tendency of the child to tire easily should be considered as of extreme importance. *Cough* in this phase of the disease is a rather rare symptom; if a child coughs the probabilities are that the cough is due to a cold and not the infection localized in the hilum glands. *Fever,* only when it is persistent, particularly in the afternoon and evening, should lead to the careful search for the cause. It should be kept in mind, however, that in younger children, temperature is more unstable than in children of older age groups.

Tuberculin tests—A positive reaction to the tuberculin test always means infection with tubercle bacilli. It does not necessarily indicate disease. We have seen many children with positive reactions, who have never developed symptoms from the infection; and it has been the experience of many investigators that often a healthy child, a child that will probably grow to full manhood without developing tuberculosis, will give a positive tuberculin reaction. Of course, no one should subject a well nourished healthy child to the "cure" just because he gave a positive tuberculin reaction. However, observations are definitely warranted in each instance. In other words, just as physical examination is not a practical measure in case-finding, the diagnosis should not be based solely on the result of tuberculin tests. There is, however, one exception. A positive test in a child under one year of age is most likely to indicate an active lesion. A positive tuberculin test in a child of that early age will indicate a rather recent exposure. During

that early age the healing capacity of a child is greatly limited and naturally there is no protective power to have the infection localized.

At present, Mantoux or intracutaneous test is considered more accurate and with that test we are able to obtain a somewhat larger number of positive reactors than was possible with the old Pirquet technique. The Vollmer patch test is also a reliable procedure in comparison to the Mantoux test. The percentage of corroboration of the two tests is rather high, and the margin of disagreement is very low. *X-ray* is absolutely indispensable in the examination of a child's chest. Without it a positive diagnosis of the primary type of tuberculosis with any degree of accuracy can not be made. A physician, no matter how good a clinician he may be, is not justified in his diagnosis of primary tuberculosis without checking his physical findings with the evidence that an x-ray film presents. Of course, there are instances in which x-ray fails to discover the infection, but these instances are rather rare and when they do occur they usually indicate a lack of sufficient calcification in the enlarged bronchial glands to give a contrast shadow.

Now in reference to the practical measures of all these discussions, we are interested mainly in two things:

1. How to find the infected child.
2. How to prevent primary infection from developing into an adult type of tuberculosis.

When tuberculosis is diagnosed in an adult it is important that all other members of the family should be examined. Children who have been in contact with that individual should have a tuberculin test and the positive reactors should be x-rayed. On the other hand, when a child is found to have tuberculosis through school surveys or in any other way, the adults in the respective family should have a thorough physical examination in order to detect the possible source of infection. Prognosis in childhood tuberculosis depends largely upon the child's reaction to the tuberculous infection and also upon exposure and dosage of the tubercle bacilli inhaled. If the source of infection has been eliminated early enough, most of the children will become adjusted to their infection unless extensive infiltration of the lungs has taken place. The majority of them, due to the high healing capacity of older children are able to withstand the infection. The infected glands usually become calcified and well walled off by fibrous tissue.

However, no matter how well healed these lesions appear to be, there is always a great danger during the period of adolescence. In a considerable number of cases of adult type of tuberculosis found in adolescents, the x-ray shows evidence of calcification referable to the preceding childhood type of infection; and it is therefore of utmost importance that boys and girls below the age of 20, who are known to have a primary type of tuberculosis, should be advised to avoid strenuous competitive exercise unless their contacts with open cases are known to have ceased in early childhood. Without this precaution there is always a possibility that excessive strain may reactivate dormant infection. It is almost impossible to prevent exposure of children to tuberculosis. While the contact of an infant is naturally limited to the immediate family and, therefore, we can do a great deal to prevent exposure by proper education and regular examination of the parents, the period of childhood is less favorable in that respect.

A school child has a much wider contact. He becomes exposed to unsuspected cases or carriers, besides his immediate family. In other words, he gets massive doses of tubercle bacilli and it is fortunate that during this period of childhood the body is better able to resist the infection than in any other period of life. And here is where we can actually help. Since the body does resist the invasion so well at this age, our aim naturally would be to promote calcification by arresting the infection at the nodular stage.

This can be best done starting with the schools, where an attempt should be made to find as many as possible of the children suffering from latent tuberculosis of the lymph nodes. These children should receive a great deal of attention as far as rest, fresh air and food are concerned. They should be kept under very close supervision by their family physicians, whose duty it is to minimize by actual care and advise the danger of developing the adult type of the disease. Every possible means should be employed to bring the children to their par, particularly those who are underweight and undernour-

ished. All sources of infection, such as infected tonsils and teeth, should be removed, nasal deformities interfering with proper breathing (deviated septum) corrected. In a great many instances the building up of the resistance will be accomplished by sufficient rest for the child, by proper, well balanced diet including rich milk and cod liver oil in any form. If such a program is carefully carried out, I believe it will aid many children in arresting the mild lymph nodes disease that they already have. This, in turn, with the proper advice and education they receive from the family physician will prevent severe forms of adult tuberculosis from developing.

It has been said that "for tuberculosis we prescribe not medicine, but a mode of life." By realizing that, our task will be greatly facilitated.

Glenn Dale Sanatorium.

Allergy in Tuberculosis

J. A. RUDOLPH, M.D.
Columbus, Ohio

When contact with a foreign protein (pollen, vegetable, animal or bacterial) that has penetrated the tissues is established, the first, ordinary or normergic response of the body varies from a mild reaction to a severe inflammation, depending upon the inherent toxicity of the agent and upon the quantity. After an incubation period, the body is then sensitized to the foreign substance, and upon repeated exposure to it the inflammatory response differs from the original reaction in that the onset is acute, the course more violent, and the resolution of the process slower. Hyperergic reactions of this sort, analogous in many respects to conditioned reflexes, occur not only in the clinical allergies but also in other forms of anaphylaxis, in idiosyncrasy, and in immunity.[1] Von Pirquet[2] in discussing the phenomena which he had included under "allergy," said as follows: "We rightly use the word 'allergy' from *ergia,* reaction, and *allos,* altered, to mean a changed reactivity as a clinical conception without being prejudiced by the bacteriological, pathological or biological findings."

This clinical conception, however, was of sufficient importance to give a new interpretation to nearly all the phenomena observed, whether bacteriological, pathological, biochemical or immunological in tuberculosis, and especially in diseases in which hypersensitivity plays a part.

Pottenger[3] in 1929 wrote as follows: "Without the consideration of allergy we can no more understand the pathological changes and varied clinical manifestations in tuberculosis than we can understand its etiology without considering the tubercle bacillus."

Koch's[4] description of the cutaneous reaction to reinfection expresses the greater part of what is known today concerning the relation of sensitization to immunity in tuberculosis. In a guinea pig infected with tuberculosis for the first time by injection into the skin, the cutaneous lesion appears slowly and persists until death, but in a previously infected animal an intense inflammatory reaction associated with superficial necrosis produces an ulcer that quickly heals without infection of the neighboring lymph nodes.

It has been well established, that allergic reactions are influenced by the number and size of the sensitizing and shocking doses and by the time intervals. They vary from functional, vascular responses and hypertonus or spasm of smooth muscle to the severe, necrotizing inflammation characterizing the Arthus phenomenon. Under certain circumstances specific granulomatous lesions result. Although it is possible that all the cells in the body may be sensitized, allergic reactions are ordinarily exhibited by the arterial system, connective and lymphoid tissues, and synovial, ectodermal and entodermal membranes, whereas the parenchymatous tissues are usually spared.

According to Rossle[5-6-7] the allergic reaction may consist entirely of a focal hyperergic reaction localizing the antigenic poison as in the Arthus phenomenon; and this organ or tissue disease prevents a blood disease. If, however, the hypersensitive state is mild or the shocking dose is extremely great, regardless of local blockage by hyperergic inflam-

mation, some of the antigen spills over into the circulation, the protection of the individual is taken up by the spleen, liver and other related protective mechanisms. If still more antigen enters the blood stream, the physiological anergy of the remaining endothelium changes to hyperergy, and by the arrest of living or dead poison on the intima, various vascular granulomata result. If this last defense is finally broken through, the antigen permeates the tissues and produces different types of inflammation depending upon the type and degree of the allergy. The successful production of endocarditis, arthritis, and myocarditis resembling the lesions of rheumatism by repeated and attenuated doses of living or dead antigens may be explained on this basis.

Rossle directly observed the changes of normergic and allergic inflammation in frog mesentery preparations. Frogs previously sensitized to swine serum, upon reapplication of this foreign protein reacted with a prompt stasis of the blood much greater in extent than in the control animals, and at the margins of the poisoned area there was a reversal of capillary blood flow. In addition there was a much more intensive accumulation of fluid and leucocytes in the sensitized animals, a greater tendency to hemorrhage and finally a greater delay in the resolution of the process.

We[8] have been able to observe reversible histological changes to antigens such as ragweed and house dust in experimentally produced wheals in allergic individuals. These inflammatory reactions were similar to those seen in the well known clinical allergies, hay fever, asthma, etc., all characterized by considerable edema and infiltration with eosinophiles, plasma cells and other wandering cells. Involved mucous membranes also show evidence of hypertrophy of smooth muscle and active secretion of mucus. Kline and Young[9] observed this type of inflammation in nasal and sinus mucous membrane in twenty-nine cases. Similar observations were made by Finck,[10] Hansel,[11] Weille,[12] and Coates and Ersner[13] and are amazingly similar in spite of varying antigens. Similarly, post mortem descriptions of tissue changes in bronchial asthma, similar to the above, appear in the literature by Huber and Koessler,[14] Kountz and Alexander,[15] and Steinberg and Figley.[16]

In certain diseases such as tuberculosis, rheumatic fever, and syphilis, the allergic inflammatory tissue changes may be not only reversible as in the positive tuberculin skin reaction and slowly resolving exudative pneumonia in tuberculosis, but also of the more persistent granulomatous type as typified by the early tubercle, Aschoff body, and early gumma, produced by specific substances after the body has been properly sensitized.

For von Pirquet, hypersensitivity meant liability to symptoms, while "insensibility," abolished reactivity meant immunity from symptoms. "It has been shown," he writes, "that the symptoms of infectious diseases are not entirely due to the action of microorganisms per se, but that, in any disease, the organism itself takes an active part in the production of most symptoms by an interaction of products of its own with products derived from the infecting agent. The products by which the organism participates in the reaction are the so-called antibodies." In discussing the special application of his concept to tuberculosis, he points out that the simple antibody antigen explanation does not seem "to cover all the types of tuberculin immunity. The reason seems to be that beside the one antibody which we considered until now, other antibodies are also involved in the process."

More recent investigative work has certainly proven the tubercle bacillus to be a highly complex organism from the chemical viewpoint; and it is interesting to point out that von Pirquet was not far off when he stated that more than one antibody might play a part in the altered reactivity of tuberculosis, as the following extract from a paper by Smithburn and Sabin[17] proved: "The lipoid portion of the organisms, notably a phosphatide constituent, possesses the capacity of producing tuberculous tissue; that is, epitheloid cells and giant cells. The so-called waxes cause a proliferation of fibroblasts. The acetone soluble fat induces proliferation of all connective tissue cells and of blood and causes hemorrhage. The polysaccharide is chemiotactic for and toxic to leucocytes. The protein is probably responsible for fever and, in addition, causes a proliferation of plasma cells."

The views of Smithburn and Sabin offer a marked contrast to the less certain atti-

tude of von Pirquet. Although the problem is still very complex, their statements are based not on wish fulfillments, but on very carefully executed experiments with purified fractions of large amounts of tubercle cultures and their results have checked on repeated tests. It appears, therefore, too narrow a conception, if one limits the term allergy to just one part of the altered state of reactivity which follows tuberculous infection.

Hypersensitivity to the tuberculo-protein is not the entire picture of allergy; the change toward immunity must be included, and to limit the use of the word "allergy" to hypersensitivity is to discount other altered reactions of like clinical importance.

I share the view of Cummins[18] in that "allergy" in tuberculosis is an expression of altered reactivity in the human or animal organism resulting from infection; a new state results in which some individuals respond to a given amount of infection by becoming extremely sensitive tc tuberculin and yet may show no resistance if the infection develops into disease (e.g., many of the negro race), while others, perhaps less hypersensitive, may be highly resistant (e.g., the Hebrew race). It seems, therefore, that one of the important functions of "acquired immunity" is to protect the infected individual from the effects of hypersensitivity.

For purposes of clarity it appears that altered reactivity in humans in relation to the tubercle bacillus and its products may be divided into three stages as follows:

Stages of tuberculosis (in terms of altered reactivity to the tubercle bacillus and its products): Normergic (neutral); 2. hyperergic (hypersensitivity): (a) mild, (b) marked; 3. Immunity: (a) negative, (b) positive.

Let us consider this classification from the practical viewpoint.

Normergic

This stage is seen early after the first infection. It is noted in experimental animals during a brief period following the inoculation, by retention of health, normal gain in weight and an absence of tuberculin sensitivity. It is observed in man in the period which elapses between the earliest contact of a newly born infant with a tuberculous sensitivity; a period which may vary from weeks to years, depending upon the virulence, quantity and frequency of infection, and possibly upon "natural immunity." In von Pirquet's sense, it is the "incubation period," the period of antibody formation. Its expression in the tissues is seen in the "primary lung focus" and in the early glandular foci, small localized lesions surrounded by healthy tissue without any sign of surrounding reaction and usually healing by peripheral fibrosis and central calcification. During this stage, the tubercle bacillus on reaching the body tissue of fluids, is no more destructive than a dust particle; reacted upon by the same phagocytes and carried along the same lymphatic route as a dust particle; and finally disposed of in the same situations or filtered out in the same collections of lymphatic drainage, by which dust, like the tubercle bacillus, travels to the glands. There is this difference between the dust particle and the tubercle bacillus. The dust particle cannot multiply, although it may produce local irritative changes. The tubercle bacillus, highly resistant to enzymes, multiplies until its increase is either limited by cellular barriers or destroyed by focal bactericidal elements. At this point, the stage of normergy or neutrality passes on, first to the stage of "mild hyperergy" and later either to that of "marked hyperergy" or possibly under favorable conditions, to relative "immunity."

Hyperegic

Mild Hyperergy—The stage of mild hyperergy is that in which most of us exist. It is brought into existence by the escape from the original focus of infection into contact with our body tissues, not necessarily with the tubercle bacillus alone but with its chemical end products, which changed by the antibodies now forming, have the power to change our entire constitutional "being" with a newly acquired reactivity. This new state is quite compatible with normal health and development and its presence is only brought to light by the tuberculin test. All that this proves at present is that infection has taken place.

Von Pirquet wrote: "if we make tests on people of different ages, we find progressively with the increase in age a growing percentage of clinically healthy people who show very slight reactivity. We might assume that in these people we are confronted by a period of lessened reactivity several years after an

acute stage. "Mild hyperergy" is a state which, following reinfection, either intrinsic or extrinsic, will be followed by a marked local and systemic reaction; in other words "marked hyperergy" to tubercle bacilli and their products.

Marked Hyperergy—Krause[19] explains the symptoms of tuberculosis as follows: "Human beings pass perfectly well as long as they hold their tuberculous infection asymptomatic. But they are allergic and any discharge of sufficient focal material to a new place will render them immediately ill because of the allergic reaction that ensues promptly." Wright[20] apparently had similar thoughts twenty years earlier when he advanced his theory of "autoinoculations." He said: "Intoxication phenomena and immunizing responses, exactly similar to those which supervene upon the inoculation of bacterial vaccins, must occur whenever bacterial products or, as the case may be, bacteria, escape from localized foci of bacterial infection and pass into the circulation." This seems to be exactly what occurs in tuberculous disease. The symptoms which follow a large dose of tuberculin in a healthy but infected person, a person in the stage of "mild hyperergy," are exactly those which occur daily in the subject of active tuberculosis as the result of autoinoculations from his active foci, malaise, headache, lassitude, and fever; Koch himself described these symptoms experienced by himself after having taken an excessive dose of old tuberculin subcutaneously. This differentiates between "mild" and "marked" hyperergy. Koch, an infected but healthy person, was only proved to be "mildly" hyperergic to the products of tubercle bacilli when these were artificially introduced into his circulation from the outside. His own foci of infection were, for the purpose of autoinoculation, extinct. But, in the "markedly hyperergic" stage the infective foci in the body are not extinct but active; or, when absolute rest has been imposed, in a quiescent state easily passing back into an active state if undue bodily exertion is unwisely attempted.

Robert Koch says: "Individual cases of the disease, have often shown that a person is not at all times an equally favourable subject for the development of parasites, for it not infrequently happens that tuberculous foci which had reacted to a fair size, contract, cicatrize and heal up." This is so often seen to occur either with or without therapy, and it implies a changed reaction present in the area of the tuberculous foci, and adaptation of the related tissues and fluids by which they are permeated to the focus and its contents so that the latter does not escape or is rendered harmless by being neutralized in some way. This altered state is to be considered as a state of immunity to the tubercle bacillus and its products and leads us to a consideration of the factors which seem to operate its activity; since it is this phase that treatment is aimed at, so that it may operate at maximum speed.

Immunity

Negative Immunity—Since "negative" immunity or "anergy" is a terminal state and occurs when treatment usually fails, there is little point in discussing it.

Positive Immunity—As has been suggested elsewhere in this paper, positive immunity depends on the diminution or complete neutralization of the effects of autoinoculations. In the spontaneous cure of symptoms, this effect must be gained by surrounding and isolating tuberculous foci and rendering these areas harmless by scar tissue about them; or through neutralization by chemical or physical means or by absorption of toxic products. There is experimental evidence that both types of defense are utilized in tuberculo-immunity. The experiments which show that tuberculous foci can be rendered innocuous, are presented to the eye through microscopic examination of certain types of tuberculous foci in which it is visibly apparent that the surrounding tissues are free from inflammatory reaction.

Cummins and Weatherell[21] have shown by their experiments with intravenous infection of rabbits with large doses of human tubercle bacilli, that "neutralization of toxins" results. These investigators showed that there was a "danger" period corresponding to the development of allergic hypersensitivity, which resulted in a number of deaths in the third and fourth week after infection and during which all infected animals were very ill. In those dying, the lungs showed marked inflammatory reaction about the numerous bacterial foci resulting from intravenous injection. In

the animals regaining their health, examination of their lungs revealed almost entirely healed areas without scar tissue formation. The earlier inflammation had been overcome and the exudate absorbed even though the lung lesions were still active.

From the evidence presented it seems that, in this disease, and in many others in which chronic bacterial foci and resulting hypersensitivity play a part, the body is faced with the problem in which efforts at spontaneous cure tend to defeat themselves. In the one case, the lysis of the tubercle bacilli destroys harmful germs but liberates harmful toxins. In the other case, the cicatrization which helps by surrounding the bacteria in avascular lesions and by rendering immobile organs in which foci are present, tends also to prevent bacteriacidal and phagocytic activity of the blood on these bacilli. Though the inflammatory response of hypersensitivity may assist in extinguishing minimal reinfections from outside, it may also destroy an infected animal if a sufficient dose of tuberculin is given; and it is also the cause of the symptoms which intensify disease. Marked hyperergy or hypersensitivity in tuberculosis is just as important in the causation of symptoms in man as the tubercle bacillus itself.

In closing, it must be emphasized that the ultimate goal is in proper methods of prevention and of treatment in tuberculosis.

Preventive treatment has made great strides. Methods of treatment, however, depend on the proper conception of the stages of tuberculosis. Our methods of cure must be directed not merely to trusting those of nature but to comprehending them and so guiding them that they shall give the maximum beneficial results. Most of our successful methods of treatment aim at the prevention or diminution of autoinoculations. Rest is the basis of these methods, either local or general, which permits the processes of immunity to become prominent instead of marked hyperergy—which determines symptoms. Collapse therapy, for example, in itself is not curative; it brings about conditions in which the immune stage of tuberculosis is able to produce its cure through the rest and cessation of autoinoculations which collapse insure to the injured lung.

Summary

When contact with a foreign protein (pollen, vegetable, animal or bacterial) that has penetrated the tissues is established, the first, ordinary or normergic response of the body varies from a mild reaction to a severe inflammation, depending upon the inherent toxicity of the agent and upon the quantity. After an incubation period the body is then sensitized to the foreign substance, and upon repeated exposure to it, the inflammatory response differs from the original reaction in that the onset in acute, the course more violent, and the resolution of the process slower. Hyperergic reactions of this sort, analogous in many respects to conditioned reflexes, occur not only in the clinical allergies but also in other forms of anaphylaxis, in idiosyncrasy, and in immunity. von Pirquet in discussing the phenomena which he had included under "allergy," said as follows: "We rightly use the word 'allergy' from *ergia*, reaction and *allos*, altered, to mean a changed reactivity as a clinical conception without being prejudiced by the bacteriological, pathological or biological findings.

For purposes of clarity it appears that altered reactivity in humans in relation to the tubercle bacillus and its products may be divided into three stages as follows: Stages of tuberculosis (in terms of altered reactivity to the tubercle bacillus and its products): 1. Normergic (neutral); 2. hyperergic (hypersensitivity): (a) mild, (b) marked; 3. immunity: (a) negative, (b) positive.

601 Huntington Bank Bldg.

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PROGRAM

Seventh Annual Meeting

American College of Chest Physicians

THE STATLER HOTEL **MAY 31 - JUNE 2nd, 1941** **CLEVELAND, OHIO**

GENERAL ARRANGEMENTS

Dr. Joseph C. Placak, *Chairman*
Dr. Dean Hugh Minnis, Amherst, Ohio
Dr. John M. Tomasch, Cleveland, Ohio
Dr. Ellery P. Edwards, Cleveland, Ohio
Dr. Gary G. Bassett, Cleveland, Ohio

SCIENTIFIC PROGRAM COMMITTEE

Dr. Benjamin Goldberg, Chicago, Illinois, *Chairman*
Dr. John H. Peck, Iowa City, Iowa
Dr. J. Winthrop Peabody, Washington, D. C.
Dr. Paul H. Holinger, Chicago, Illinois

ENTERTAINMENT COMMITTEE

Dr. Louis Mark, Columbus, Ohio, *Chairman*
Dr. Edward Arnold, Canton, Ohio
Dr. John Harvey Skavlem, Cincinnati, Ohio
Dr. Samuel O. Freedlander, Cleveland, Ohio
Dr Paul William Gebauer, Cleveland, Ohio
Dr. Casper H. Benson, Columbus, Ohio
Dr. Warren C. Breidenbach, Dayton, Ohio
Dr. Paul M. Holmes, Toledo, Ohio
Dr. Robert H. Browning, Warrensville, Ohio
Dr. E. E. Kirkwood, Youngstown, Ohio.

TRANSPORTATION COMMITTEE

Dr. Robert H. Browning, *Chairman*
Dr. H. C. Schock, Cleveland, Ohio
Dr. Joseph B. Stocklen, Cleveland, Ohio

COMMITTEE ON CLINICS

Dr. Paul William Gebauer, Cleveland, Ohio
Chairman
Dr. Joseph B. Stocklen, Cleveland, Ohio
Dr. Sidney E. Wolpaw, Cleveland, Ohio
Dr. Harold Curtis, Cleveland, Ohio

COMMITTEE ON SCIENTIFIC EXHIBITS

Dr. Jack Appel, Cleveland, Ohio, *Chairman*
Dr. Dean H. Minnis, Amherst, Ohio
Dr. H. C. Schock, Cleveland, Ohio
Dr. Harold Curtis, Cleveland, Ohio

SURGICAL SECTION

Dr. S. O. Freedlander, Cleveland, Ohio, *Chairman*

MEDICAL SECTION

Dr. Raymond C. McKay, Cleveland, Ohio, *Chairman*

SATURDAY, MAY 31, 1941

Registration: Mezzanine, Euclid Ball Room
9:30 A. M.—SCIENTIFIC SESSION

SALE MODERNE

Dr. Samuel A. Freedlander, Cleveland, Ohio
Chairman

"Visualization of Cavities in Post-Thoracoplasty Lungs"
Dr. Jerome L. Leon
Dr. Henry Green
Dr. Charles A. Serbst
New York, N. Y.
Discussion to be opened by: Dr. Paul Turner
Louisville, Ky.

"Foreseeing and Forestalling Tuberculosis"
Dr. Wm. Ogden
Dr. G. C. Anglin
Dr. W. C. Kruger
Toronto, Canada
Discussion to be opened by: Dr. Edward P. Eglee
New York, N. Y.
Dr. Frederick A. Slyfield
Seattle, Wash.

"The Management of Fluids Complicating Pneumothorax"
Dr. Dean B. Cole
Dr. Walter L. Nalls
Richmond, Virginia
Discussion to be opened by: Dr. C. Howard Marcy
Pittsburgh, Pa.
Dr. William Devitt
Allenwood, Pa.

12:00 P. M.—"INFORMATION PLEASE"

LUNCHEON

EUCLID BALLROOM

Dr. Benjamin Goldberg,
Chicago, Ill.
Chairman

Surgery
Dr. Richard Overholt
Boston, Mass.
Bronchology
Dr. Louis H. Clerf
Philadelphia, Pa.
Medicine
Dr. Edgar Mayer
New York, N. Y.
Surgery
Dr. Ralph C. Matson
Portland, Ore.
Physiology
Dr. Harold Green
Cleveland, Ohio
Roentgenology
Dr. Joseph Post
Philadelphia, Pa.

SATURDAY, MAY 31, 1941

2:00 P. M.—SCIENTIFIC SESSION

SALE MODERNE

Dr. Raymond C. McKay,
Cleveland, Ohio
Chairman

"Monaldi Procedure"
Dr. Edward Kupka
Olive View, Calif.
Discussion to be opened by: Dr. Milton Lerner
Oakdale, Iowa

"Pregnancy and Tuberculosis"
Dr. Frederick H. Falls
Chicago, Ill.
Discussion to be opened by:
Dr. Fred M. Meixner
Peoria, Ill.
Dr. Wm. C. Voorsanger
San Francisco, Calif.

"A New Extrapleural Pack in the Treatment of Pulmonary Tuberculosis"
Dr. Ralph C. Matson
Portland, Oregon
Discussion to be opened by: Dr. John W. Stacey
Tucson, Ariz.
Dr. Edward J. O'Brien
Detriot, Mich.

3:00 P. M. Board of Regents Meeting—Tavern Room
6:00 P. M., Cocktail Party
7:00 P. M., Dinner Meeting of the House of Governors and Boards of Regents (formal). (Open to all Fellows and Associates of the College).
8:00 P. M., Administrative Session

Address of Welcome
Dr. Joseph C. Placak
Cleveland, Ohio

Presidential Address
Dr. John H. Peck
Oakdale, Iowa

Report of the Secretary-Treasurer
Dr. Paul H. Holinger
Chicago, Ill.

Report of the Board of Regents
Dr. Frank Walton Burge
Philadelphia, Pennsylvania
Chairman, Board of Regents

Report of Committees
Military Affairs Committee
Dr. Chas. M. Hendricks
El Paso, Texas
Statistical Committee
Dr. H. B. Freilich
Chicago, Illinois

Committee for the Advancement of Undergraduate Teaching in Medical Schools
Dr. E. W. Hayes
Monrovia, California

Committee for the Advancement of Scientific Programs in Organized Medicine
Dr. Ralph C. Matson
Portland, Oregon

Committee on Sanatorium Standards
Dr. R. H. Runde
Mt. Vernon, Missouri

Committee for the Advancement of Tuberculosis Organization in Medicine
Dr. Paul A. Turner
Louisville, Kentucky

Sanatorium Committee
Dr. Harry C. Warren
San Francisco, California

Constitution and By-Laws Committee
Dr. J. Winthrop Peabody
Washington, D. C.

Membership Committee
Dr. Jay A. Mayer, *Chairman*
Minneapolis, Minnesota

Report of Nominating Committee

Election of Officers

Address of Incoming President
Dr. Benjamin Goldberg
Chicago, Illinois

COMMITTEE MEETINGS

10:00 A. M. Statistical Committee ..... Parlor A
10:00 A. M. Committee on Sanatorium Standards ..... Parlor B
10:00 A. M. Military Affairs Committee ..... Parlor D
11:00 A. M. Committee for the Advancement of Undergraduate Teaching in Medical Schools ..... Parlor A
11:00 A. M. Committee for the Advancement of Scientific Programs in Organized Medicine ..... Parlor B
11:00 A. M. Committee for the Advancement of Tuberculosis Organization of Medicine ..... Parlor D
2:00 P. M. Constitution and By-Laws Committee ..... Parlor A
2:00 P. M. Nominating Committee ..... Parlor B
2:00 P. M. Sanatorium Committee ..... Parlor D

SUNDAY JUNE 1, 1941

10:00 A. M.—SCIENTIFIC SESSION

SALE MODERNE

Dr. Warren C. Breidenbach,
Dayton, Ohio
Chairman

"Bronchial Obstruction as an Etiologic and Perpetuating Factor in Empyema"
Dr. Chevalier L. Jackson
Philadelphia, Pa.
Discussion to be opened by: Dr. John S. Knight
Kansas City, Mo.
Dr. Paul H. Holinger
Chicago, Ill.

"The Management of Genito-Urinary Tuberculosis"
Dr. Monroe E. Greenberger
Dr. Arthur J. Greenberger
New York, N. Y.
Discussion to be opened by: Dr. Frederick Lieberthal
Chicago, Ill.

12:00 P. M. LUNCHEON—ROUND TABLE DISCUSSION—ROOM 345

Committee for the Advancement of Undergraduate Teaching in Medical Schools
Dr. E. W. Hayes, *Chairman*
Monrovia, California

2:00 P. M.—SCIENTIFIC SESSION

SALE MODERNE

Dr. John Skavlem,
Cincinnati, Ohio
Chairman

"Some of the Newer Concepts of the Tubercle Bacillus and Its Chemical Susceptibilities"
Dr. Ralph R. Mellon
Pittsburgh, Pa.
Discussion to be opened by: Dr. Julius Wilson
New Orleans, La.
Dr. Emil Bogen
Olive View, Calif.

"The Clinical Aspects of Cancer of the Lungs"
Dr. George G. Ornstein
New York, N. Y.
Discussion to be opened by: Dr. Henry F. Carman
Dallas, Texas
Dr. George G. Gilbert
Colorado Springs, Colo.

"The Present Day Treatment of Epyema, a Report of over 500 Cases"
Dr. Willard Van Hazel
Chicago, Ill.

Discussion to be opened by: Dr. Victor S. Randolph
Phoenix, Ariz.
Dr. Hans E. Schiffbauer
Los Angeles, Calif.
"Fungous Diseases of the Lungs"
Dr. Arthur Q. Penta
Schenectady, N. Y.
Discussion to be opened by: Dr. Mathew Jay Flipse
Miami Florida
Dr. Francis M. Pottenger, Sr.
Monrovia, Cal.
7:00 P. M. Banquet—EUCLID BALLROOM (formal)
Dr. Champ H. Holmes
Atlanta, Georgia
Toastmaster
Guest Speaker
Dr. Louis Dublin, Vice President
Metropolitan Life Insurance Company
New York, N. Y.
Subject: "The Next Ten Years in Tuberculosis: A Forecast."

MONDAY, JUNE 2, 1941

9:30 A. M. *Clinics*—Cleveland City Hospital
Dr. Paul Gebauer,
Cleveland, Ohio
Chairman
Luncheon—Hollenden Hotel—Sanatorium Committee
Dr. Harry C. Warren,
San Francisco, Calif.
Chairman
Luncheon—Hollenden Hotel—Board of Regents
Dr. Frank Walton Burge,
Philadelphia, Pa.
Chairman
2:00 P. M.—*Clinics*—Cleveland City Hospital
Dr. Paul Gebauer,
Cleveland, Ohio
Chairman
7:00 P. M.—Smoker and Dinner—Hollenden Hotel (informal)
Dr. Louis Mark,
Columbus, Ohio
Chairman, Entertainment Committee

STATE CHAPTER MEETINGS

8:30 P. M.—Organization Meeting of the Ohio State Chapter of the College
Speaker: Dr. J. Winthrop Peabody,
Washington, D. C.
8:30 P. M.—Annual Meeting of the New York State Chapter of the College
Presiding: Dr. Edgar Mayer, President,
New York, N. Y.
Speaker: Dr. Benjamin Goldberg,
Chicago Ill.

Organization News

COMING MEETINGS

AMERICAN COLLEGE OF CHEST PHYSICIANS, Cleveland, *May 31 - June 2*. Dr. Paul H. Holinger, 500 North Dearborn Street, Chicago, Secretary.

TEXAS CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS, Fort Worth, May 13th. Dr. Orville E. Egbert, First National Bank Building, El Paso, Governor.

ILLINOIS CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS, Chicago, May 20th. Dr. Robert K. Campbell, St. Johns Sanitarium, Springfield, Governor.

NEW JERSEY CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS, Atlantic City, May 21st. Dr. Chas. I. Silk, 236 High Street, Perth Amboy, Secretary.

NEW YORK STATE CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS, Cleveland, June 2nd. Dr. Arthur Q. Penta, 713 Union Street, Schenectady, Secretary.

OHIO STATE CHAPTER, AMERICAN COLLEGE OF CHEST PHYSICIANS, Cleveland, June 2nd. Dr. Louis Mark, 677 North High Street, Columbus, Governor.

QUESTIONS PLEASE

Because of the popular reception accorded to the "Information Please" luncheon meeting held last year at New York City, it has been decided to hold another of these informal gatherings at the Cleveland Meeting of the College. The following experts have been invited to answer your questions:

Surgery: Dr. Richard Overholt, Brookline, Massachusetts and Dr. Ralph C. Matson, Portland, Oregon.

Medicine: Dr. Edgar Mayer, New York City, New York.

Bronchology: Dr. Louis H. Clerf, Philadelphia, Pennsylvania.

Physiology: Dr. Harold Green, Cleveland, Ohio.

Radiology: Dr. Joseph W. Post, Philadelphia, Pennsylvania.

An invitation is extended to the members of the College and to the readers of this Journal to send their questions in on subjects related to diseases of the chest to Dr. Benjamin Goldberg, Chairman of the Scientific Program of the College for the Cleveland Meeting, 58 East Washington Street, Chicago, Illinois.

The luncheon meeting will be held at the Statler Hotel, Cleveland, on May 31st.

TEXAS CHAPTER TO ORGANIZE

The Texas Chapter of the American College of Chest Physicians will be organized at the meeting of the State Medical Association at Fort Worth, Texas, on May 13th. There will be a dinner in the Oak Room of the Texas Hotel at 6:00 p. m., followed by a scientific program and organization meeting. Dr. R. G. McCorkle of San Antonio, Texas, will read

a paper on "Fungus Disease of the Lung." The paper will be discussed by Dr. Abbe Ledbetter, Houston, Texas and by Dr. Leslie Smith, El Paso, Texas. Dr. Orville E. Egbert, El Paso, Texas, Governor of the College for Texas will be the presiding officer for the meeting. The State Medical Association of Texas will meet at Fort Worth, May 12-15th.

ILLINOIS CHAPTER TO HOLD ANNUAL MEETING

The annual meeting of the Illinois Chapter of the American College of Chest Physicians will be held at the Palmer House, Chicago, on May 20th. This meeting will be held in conjunction with the annual meeting of the Illinois State Medical Society,

Chicago, May 20-23rd.

The Arrangements Committee reports that there will be a dinner served at 6:00 p. m., this to be followed by the main speaker, Dr. Richard Davison, Chicago, Illinois, whose subject will be "Extrapleural Pneumothorax." Dr. Davison is the chief thoracic surgeon at the Municipal Tuberculosis Sanitarium. This will be followed by a discussion of the paper from the floor. After a short recess, the business meeting of the State Chapter will be held and election of officers will take place. Dr. Robert B. Campbell, Springfield, Illinois, President of the Illinois Chapter of the College will preside.

For reservations and for further information regarding meeting, address the Executive Offices of the College, 500 North Dearborn Street, Chicago, Illinois.

MISSOURI CHAPTER OF COLLEGE ORGANIZES

The Missouri State Chapter of the American College of Chest Physicians was organized at a meeting held at the Jefferson Hotel, St. Louis, Missouri, on April 29th. Dr. H. I. Spector, St. Louis, Governor of the College for Missouri, was the presiding officer. The Missouri State Medical Society held its annual meeting at St. Louis, April 28-30th.

NEW JERSEY CHAPTER TO HOLD ANNUAL MEETING

The annual meeting of the New Jersey Chapter of the American College of Chest Physicians will be held at Atlantic City, New Jersey, on May 21st. The Chapter will hold a breakfast meeting at which time the progress of the Chapter for the past year will be reviewed, and election of officers for the ensuing year will take place. Dr. Frank Walton Burge, Philadelphia, Chairman of the Board of Regents of the College, will be the guest speaker. The New Jersey Chapter of the College will meet in conjunction with the New Jersey State Medical Society, which meets at Atlantic City, May 20-22nd. Dr. Martin H. Collier, Grenloch, New Jersey, president of the New Jersey Chapter of the College will preside at the meeting. For further particulars, address Dr. Charles I. Silk, Secretary, New Jersey Chapter, American College of Chest Physicians, 236 High Street, Perth Amboy, New Jersey.

OHIO CHAPTER TO ORGANIZE

The Ohio members of the American College of Chest Physicians will meet to form a State Chapter of the College at Cleveland on June 2nd. Ohio, with 53 Fellows and Associates in the College, is the host state to the annual meeting of the American College of Chest Physicians to be held at Cleveland, May 31-June 2nd. Following a dinner, a business meeting will be held by the Ohio members for the purpose of ratifying a Constitution and By-Laws for the State Chapter and to elect officers. The meeting will be addressed by the officers of the national organization. For further information concerning this meeting, address Dr. Louis Mark, Governor of the American College of Chest Physicians, 677 North High Street, Columbus, Ohio.

NEW YORK STATE CHAPTER TO MEET AT CLEVELAND

The New York State Chapter of the American College of Chest Physicians will meet this year at Cleveland, Ohio, in connection with the annual meeting of the College. This meeting will be held on Monday night, June 2nd. A report of the progress made during the past year will be given and the election of officers for the ensuing year will be held. Dr. Benjamin Goldberg, Chicago, president elect of the College will be the guest speaker. Dr. Edgar Mayer, New York City, president of the New York State Chapter of the College, will preside at the meeting. For further information, address Dr. Arthur Q. Penta, secretary of the New York State Chapter of the American College of Chest Physicians, 713 Union Street, Schenectady, New York.

ILLINOIS CHAPTER OF THE COLLEGE MEETS

The Illinois State Chapter of the American College of Chest Physicians met at the Fox River Tuberculosis Sanatorium, Batavia, Illinois, on March 24th. Following an inspection of the sanatorium, the members were the guests of Dr. Jacob J. Mendelsohn, Medical Director of the institution, at a delightful chicken dinner. The following program was presented after the dinner:

1. *Progress of the College from a National Viewpoint,*

   Murray Kornfeld, Chicago; Executive Secretary, American College of Chest

Physicians.

2. *Hexylresorcinal in the Treatment of Tuberculous Empyema,*
   Jacob J. Mendelsohn, M.D., Chicago.
   Discussion by:
   Otto L. Bettag, M.D., Pontiac,
   W. H. Watterson, M.D., LaGrange,
   Otto C. Schlack, M.D., Oak Forest,
   Arthur S. Webb, M.D., Wheaton.
3. *What is the Prognostic Value of the Tuberculin Test?*
   W. H. Watterson, M.D., LaGrange.
4. *What is Chronic Psuedo-Membranous Bronchitis?*
   Paul H. Holinger, M.D., Chicago.
5. *Has the Surgical Management of Pulmonary Tuberculosis Influenced in any way the Incidence and Pathology of Laryngeal Tuberculosis?*
   Jacob J. Mendelsohn, M.D., Chicago.
6. *When is Pregnancy an Indication for Therapeutic Abortion in a Patient with Active Pulmonary Tuberculosis?*
   Fred M. Meixner, M.D., Peoria.

Dr. Robert K. Campbell, Springfield, President of the Illinois Chapter and Governor of the College for Illinois presided. The following members and guests attended the meeting:

Hugh A. Beam, M.D., Moline; R. C. Benkendorf, M.D., Bushnell; Otto L. Bettag, M.D., Pontiac; Robert K. Campbell, M.D., Springfield; D. A. DePinto, M.D., Chicago; Paul H. Holinger, M.D., Chicago; C. M. Jack, M.D., Decatur; Minas Joannides, M.D., Chicago; Aaron Keinigsberg, M.D., Chicago; Bernard Klein, M.D., Joliet; S. Klein, M.D., Aurora; Murray Kornfeld, Chicago; A. J. Levy, M.D., Gilman; J. L. Marks, M.D., Chicago; H. E. Mehmert, M.D., Chicago; *Fred M. Meixner, M.D., Peoria; Jacob J. Mendelsohn, M.D., Chicago; Clare Miller, M.D., Quincy; Louis J. Miller, M.D., Chicago; A. L. Nickerson, M.D., Kankakee; George Thomas Palmer, M.D., Springfield; Otto C. Schlack, M.D., Oak Forest; Ethelyme Sullivan, M.D., East St. Louis; Darrell H. Trumpe, M.D., Springfield; W. H. Watterson, M.D., La Grange; Arthur S. Webb, M.D., Wheaton; H. C. Worthington, M.D., Oak Forest.

The next meeting of the Illinois State Chapter of the American College of Chest Physicians will be held at the Palmer House, Chicago, on May 20th. This meeting will be the regular annual meeting of the State Chapter and, in addition to a scientific program, an election of officers will be held. The following Committee on Nominations has been appointed: Dr. Fred M. Meixner, Peoria, *Chairman;* Dr. Minas Joannides, Chicago; and Dr. Arthur S. Webb, Wheaton. The Illinois State Medical Society will meet at the Palmer House, Chicago, May 20 - 22nd.

* Regent for District No. 6.

PRESIDENT ADDRESSES MEETING AT ST. LOUIS

Dr. John H. Peck, Oakdale, Iowa, president of the American College of Chest Physicians, addressed the joint meeting of the St. Louis Trudeau Society and the St. Louis County Medical Society, at St. Louis, Missouri, on April 8th. Dr. Peck spoke on "The Evaluation of the Modern Method of Diagnosis and Treatment of Pulmonary Tuberculosis." Dr. Evarts A. Graham, St. Louis, Missouri, discussed the paper.

Dr. Arthur Q. Penta, Schenectady, New York, Secretary of the New York State Chapter of the American College of Chest Physicians recently was the guest speaker before the Staff of St. Francis Hospital, Miami Beach, Florida. Dr. Penta spoke on the use of iodized oil in the treatment of chronic bronchitis, bronchiectasis, and bronchial asthma.

DR. PAINTER ELECTED PRESIDENT

Dr. J. Carl Painter, Dubuque, Iowa, Governor of the American College of Chest Physicians for the State of Iowa was elected president of the Iowa Clinical Medical Society at a meeting held at Iowa City, Iowa, on April 5th.

COLLEGE GOVERNORS CONDUCT LECTURE TOUR

Dr. John F. Allen, Omaha, Nebraska, Governor of the American College of Chest Physicians for Nebraska and Dr. H. I. Spector, St. Louis, Missouri, Governor of the College for Missouri, were the guest speakers on a lecture tour of the State of Kansas conducted under the auspices of the Kansas Tuberculosis Association. Both speakers were scheduled to address a number of meetings in various parts of the State of Kansas during a week's tour.

COMMITTEE TO STUDY CONSTITUTION AND BY-LAWS APPOINTED

Dr. John H. Peck, Oakdale, Iowa, president of the American College of Chest Physicians has appointed the following committee to make a study of the Constitution and By-Laws of the College and to bring in recommendations for a revision of the present Constitution and By-Laws: Dr. J. Winthrop Peabody, Washington, D. C., *Chairman;* Dr. Frank Walton Burge, Philadelphia, Pennsylvania; Dr. E. W. Hayes, Monrovia, California; Dr. Robert B. Homan, Jr., El Paso, Texas; and Dr. Paul H. Holinger, Chicago, Illinois, *Secretary.*

Dr. Peck in his letter to the members of the committee stated: "The College has shown such gratifying growth and development during its relatively short period of existance that some of the Fellows have suggested that our Constitution and By-Laws be re-written. The experience of the past six years has necessitated several amendments and now the entire code should perhaps be clarified by complete revision, so that it will more satisfactorily serve the needs of a constantly expanding organization."

The Committee will bring in their report at the 1941 Meeting of the College to be held at the Hotel Statler, Cleveland, May 31-June 2nd, 1941.

NOTICE

Effective June 15th, 1941, Written Examinations will be required for Fellowship. Effective January 1st, 1942, the Fellowship Fee will be $100.00.

OBITUARY

Jacob Paul Frantz, M.D.

1885-1941

The State of Pennsylvania and the American College of Chest Physicians lost one of their most valuable leaders in the fight against tuberculosis at 1:05 a. m. on April 8, 1941. Dr. Frantz' death was caused by a heart condition, complicated by a kidney ailment, which had confined him to bed since January.

Dr. Frantz was born in Lancaster, Pennsylvania and spent his early boyhood on a farm. He attended Franklin and Marshall College, graduating in 1906. From there, he went directly to the Medical School of the University of Pennsylvania, receiving his M.D. degree in 1910.

After practicing for 5 years in Philadelphia, Dr. Frantz came to Clearfield, where he opened that city's first chest clinic and where he remained until his death.

While in Clearfield, Dr. Frantz organized the Clearfield County Tuberculosis Society and was its President at the time of his death. His efforts for civic betterment, both medical and non-medical, have endeared him to the hearts of the people of Clearfield.

Dr. Frantz was the first Governor of Pennsylvania for the American College of Chest Physicians and served on the Committee on Tuberculosis of the Medical Society of the State of Pennsylvania.

He is survived by his wife and five children.